protective agents comprising ginsenoside Rb_1"). As the results, we have found that ginsenoside Rb_1, at such a markedly low concentration range never reported in the world as 1 fg/ml to 100 fg/ml, suppresses apoptosis-like nerve cell death by increasing the expression of a cell death-suppressing gene product Bcl-X_L.

Namely we have found that ginsenoside Rb_1 is the only one non-peptidic stimulator of Bcl-X_L expression in the world. Although ginsenoside Rb_1 at the concentration of 100 fg/ml showed a slightly suppressive action on the formation of lipid peroxides, no such effect was observed at a lower concentration range. Consequently, the hypothesis heretofore presented in relation to the action mechanism of ginsenoside Rb_1, namely the hypothesis that ginsenoside Rb_1 decreases cell membrane lipid peroxides as a result of erasing hydroxyl radicals to protect nerve cells, was found inappropriate.

Further, we have found that intravenous administration of ginsenoside Rb_1 exhibits unexpectedly a superior suppressive action against cerebral infarction and ameliorates infarction-induced place navigation disability (JP98/365560, PCT/JP99/02550: "Brain cell or nerve cell-protective agents comprising ginsenoside Rb_1").

However, if vascular regeneration and reconstruction do not occur in the ischemic brain tissue, i.e. the ischemic penumbra, which is relieved of entering cerebral infarction as

a result of administering ginsenoside Rb_1, even after termination of the intravenous administration of ginsenoside Rb_1, there is a high possibility that new injuries to the brain may appear later in the same region after termination of the administration of ginsenoside Rb_1. In addition, even if the primary infarcted lesion of the cerebral cortex is ameliorated by the intravenous administration of ginsenoside Rb_1, unless the secondary degeneration in the thalamus which has close synaptic connections with the cerebral cortex, is inhibited, the effect and efficacy of intravenously administered ginsenoside Rb_1 may not be fully elicited.

We have found that the intravenous administration of ginsenoside Rb_1 promotes regeneration and/or reconstruction of the vascular networks in the ischemic penumbra and have completed the present invention. We have also found that the intravenous administration of ginsenoside Rb_1 suppresses the secondary degeneration of the thalamus which is generated after cerebrocortical infarction, and the secondary degeneration of the nervous tissues which occurs after spinal cord injury, and have completed the present invention.

Disclosure of the Invention

An object of the present invention is to provide pharmaceutical compositions or products promoting, in a superior manner, cerebrovascular regeneration and

reconstruction by an intravenous administration after cerebral apoplexy as well as protecting the injured or damaged brain for a long period by inhibiting the secondary degeneration of the nervous tissues.

Another object of the present invention is to provide efficacious preparations for administration comprising ginsenoside Rb_1 or its salt useful as the promoters of cerebrovascular regeneration and/or reconstruction and as the inhibitors of secondary nervous tissue degeneration. More particularly, the present invention provides the pharmaceutical compositions comprising ginsenoside Rb_1 or its salt for promoting the cerebrovascular regeneration and/or reconstruction, or the pharmaceutical compositions comprising ginsenoside Rb_1 or its salt which inhibit the secondary nervous tissue degeneration. Further object of the present invention is to provide the preparations comprising ginsenoside Rb_1 or its salt for intravenous administration useful for long term therapy, prevention or treatment of cerebral or nervous diseases.

Brief Description of Drawing

Fig. 1 shows the results of water maze tests using rats intravenously infused with ginsenoside Rb_1. The left drawing in Fig. 1 shows the results of water maze tests at the second week after MCA permanent occlusion and the right drawing shows the results of water maze tests at the fourth week after MCA occlusion.